SUNLIGHT AND AGE-RELATED EYE DISEASE

Richard W. Young, PhD
Los Angeles, California

Within 50 years, if current trends continue, 50 million elderly Americans will suffer visual impairment from macular degeneration or cataract. However, available evidence indicates that this impending crisis of visual health can be minimized by a simple, safe, inexpensive, and practical means of prevention. Cataract and macular degeneration are the ultimate consequences of normal aging, a lifelong process of deterioration. Three major causes of ocular deterioration have been identified: oxygen, heat, and solar radiation. Among these, the radiation hazard is readily accessible to human intervention. The lens is damaged by ultraviolet radiation in sunlight, whereas the retina can be harmed by high-energy visible radiation (the "violet and blue"). Use of sunglasses that block all ultraviolet radiation and severely attenuate high-energy visible radiation will slow the pace of ocular deterioration and delay the onset of age-related disease, thereby reducing its prevalence. A 20-year delay would practically eliminate these diseases as significant causes of visual impairment in the United States. (*J Natl Med Assoc.* 1992;84:353-358.)

Key words • cataracts • macular degeneration • visual impairment • elderly

One measure of the success of the health professions is how long we live. At the turn of the century, the average life expectancy in the United States was about 50 years. Now it is nearly 75 and still rising. Within 50 years, one out of four Americans will be older than 65.

The quantity of life has been dramatically extended, but the quality of life in later years commonly deteriorates. Although our survival has been prolonged, the capacity to prevent or treat disorders of old age has not kept pace. As a result, ailments associated with senescence have steadily increased in significance. Ocular disease is no exception—visual impairment is now primarily an affliction of the elderly.

Chief among these age-related eye diseases are cataracts, opacifications of the lens that prevent light from reaching the retina, and macular degeneration, a loss of central vision resulting from the death of rods and cones.

Increasing longevity raises the likelihood of suffering a loss of eyesight from one of these diseases. Between the ages of 65 and 75, there is a 10% chance of losing vision from retinal degeneration, and a 25% probability that visual acuity will be reduced by cataract. If individuals live past the age of 75, they have a 30% chance of visual impairment from retinal degeneration and a 50% probability of diminished vision from cataract. More than half of us can look forward to spending our later years with reduced visual capacity.

Based on current prevalence rates and population estimates for the future, it is projected that in 50 years 15 to 20 million elderly Americans will suffer from macular degeneration, and 25 to 36 million more will have their vision reduced by cataract. Currently, more than 1.5 million cataract operations are performed annually in this country at a medical and surgical cost of more than $7 billion. At the present rate of increase, in 50 years the number will approach 10 million operations annually at a cost in current dollars of $50 billion.[1]

Cataract surgery is one of the great achievements of ophthalmology as it restores useful vision for most patients. In the long run, however, it cannot stem the tide of the impending visual health catastrophe.

From the Department of Anatomy and Cell Biology and the Jules Stein Eye Institute, UCLA Center for Health Sciences, Los Angeles, California. Requests for reprints should be addressed to Dr Richard W. Young, Dept of Anatomy, UCLA Medical School, Los Angeles, CA 90024-1763.

Some developed nations might be able to divert sufficient resources for an expansion of cataract surgery to the magnitude that will be needed. However, cataracts are a global problem; it is humanity's major cause of blindness. Worldwide, more than 40 million people are now afflicted. Unfortunately, mounting a massive surgical effort against cataract in many parts of the world is clearly impractical. Furthermore, no medical or surgical treatment can resurrect visual cells whose death produces the visual loss of macular degeneration.

Another possibility for averting the coming crisis in visual health of the elderly is medical treatment. Unfortunately, this approach also seems to be impractical. Cataract and macular degeneration result from a progressive deterioration of the normal cellular and molecular structure of the lens and retina. Any attempt to restore the native state by introducing drugs into the system is more likely to cause additional ocular abnormalities (and produce deleterious side effects in other tissues) than it is to repair damage that has already occurred. There is currently no support for the concept that these diseases might be cured by pharmacological intervention.

With surgery and medical therapy offering limited hope, any opportunity that might offer the possibility of preventing the visual impairment of old age deserves serious consideration. Such an opportunity now exists. It appears that the development of both diseases could be impacted on simultaneously by a scientifically sound means of prevention that has the advantages of being simple, safe, inexpensive, practical, and available.

AGING AND AGE-RELATED EYE DISEASE

Age-related cataract and macular degeneration are two separate diseases that affect distinctive parts of the eye and have different clinical and pathological features. The cells and molecules that are affected in the two conditions are almost totally dissimilar. Nevertheless, recent research has revealed that these diseases have several fundamental common characteristics, and both are the ultimate outcome of the normal process of aging, which affects all eyes.[1-3]

Age-Related Cataract

The lens, surrounded by its capsule, is the only pure population of cells in the body. The vast majority of these cells are dead. There is no mechanism for eliminating damage that may occur in this mass of protein-filled cell carcasses.

The major features of age-related cataract are a progressive deterioration of the native molecules of the lens involving oxidation, aggregation, insolubilization, and nuclear pigmentation. Racemization, deamidation, glycosylation, and partial hydrolysis of proteins are also observed. Ultimately, the aberrant discontinuities in lens structure attain dimensions sufficient to reduce transparency by scattering light. Pigmentation of the lens nucleus may also interfere with transparency by absorbing light.

These changes are not unique to the cataractous lens in elderly persons. Indeed, they are detectable in lesser degree early in life, even in the eyes of children. They are part of an unremitting deterioration of the living structure referred to as aging. The name of the condition is changed from "aging" to "cataract" when the deterioration begins to interfere with vision, but at the cellular and molecular level, there is no discontinuity whatsoever.[1]

Age-Related Macular Degeneration

The major feature of this disease is the death of centrally situated rods and cones, resulting from a degeneration of the associated pigment epithelium. The demise of the pigment epithelium, in turn, can be traced to the intracellular accumulation of lipofuscin. This material consists of molecules that have been damaged so severely that the cells' native enzymes cannot degrade them. Here also the onset of deterioration can be detected in the eyes of young children. Lipofuscin begins to accumulate in the pigment epithelium by the second decade of life. With increasing age, the cells gradually become engorged with these sacs of useless, damaged molecules. This is not macular degeneration; this is normal aging. These changes occur in the eyes of all individuals.

In many eyes, by the sixth or seventh decade of life, aberrant excretions begin to occur along the basal surface of the pigment epithelial cells, producing mechanical distortion, interfering with metabolite exchange, and reducing adherence of the cells to Bruch's membrane. Finally, in some eyes, the transition from aging to disease occurs. Visual function begins to be disturbed. Vision is lost when photoreceptor cells die because of the degeneration of the pigment epithelium or the disruptive consequences of blood vessels that invade the anomalous deposits extruded from the deteriorating epithelium.[2]

Thus, there is an important common feature of these two age-related ocular afflictions. In both, there is a continuum of aging and disease. They are earlier and later stages of the same lifelong process of deteriora-

tion. The loss of vision is an affliction of the elderly, but the natural history of the disease begins early in life. With each passing year, the probability rises that the progressive deterioration will interfere with vision, thereby signaling the transition from aging to disease.

WHAT ARE THE CAUSES OF OCULAR AGING?

The gradual deterioration of the native state we call aging (or senescence) is an inevitable consequence of the basic laws of nature, expressed in the entropy principle. However, although decay is ineluctable, the rate at which it occurs is not. The rapidity of the aging process is flexible and depends on the effects of several different factors—some inherent in the body, some occurring outside it—that accelerate or retard the rate of deterioration.[1] Some of these factors are accessible to human intervention.

Now we arrive at a key question: What are the agents that produce the deterioration? What are the major causal factors whose scientifically documented effects can produce the cellular and molecular changes that characterize the degeneration of the lens and the outer layers of the center of the retina? Three major causal factors have been identified. Two of these primary causes of deterioration affect all parts of the body, and both are deleterious side effects of fundamental life processes.

The first is oxygen. Oxygen and its by-products can alter native molecules by removing electrons and hydrogen atoms, setting off chains of free-radical reactions that can disrupt any biological molecule in their path. All cells are at risk. The side effects of oxygen metabolism play a major role in the deterioration of both the lens and retina, just as they do in other parts of the body.

The second major cause of aging is heat—the energy of molecular movement. Heat is a source of deteriorative change throughout the body. Local concentrations of kinetic energy, transferred by molecular collision, occasionally exceed the threshold of molecular bonds, damaging the native state. This also occurs in the lens and retina, where raising the temperature of the tissue hastens the rate of deterioration.

The third causal factor is radiation or sunlight. Solar radiation causes damage in all parts of the body exposed to it—namely, the skin and the eyes. Solar radiation contributes to ocular deterioration in three ways. First, solar radiation exacerbates thermal damage. Sunlight is pure radiant energy. When this energy is absorbed in the lens or retina, it is converted to heat energy, thereby increasing the rate of thermal damage. Second, solar radiation exacerbates oxidative damage. It does this by producing excited-state oxygen molecules—oxygen molecules with extra energy that can set off free-radical chain reactions capable of damaging all types of molecules in both the lens and retina. Third, the energy of solar radiation can damage molecules by its own direct (photochemical) effects.

A substantial body of experimental and laboratory evidence supports the conclusion that solar radiation, acting in concert with internal heat and oxygen, can account for all of the major features of the age-related deterioration of both the lens and the retina in impressive molecular detail. Even the retinal location of macular degeneration can be accounted for by the radiation factor.[3] Whenever experimental animals are kept in a bright-light environment, the greatest cellular and molecular damage is always located in the outer layers of the center of the retina, in the photoreceptor cells and the pigment epithelium. This is precisely the site of lesion in macular degeneration. Clinical observations are also accounted for, such as the well-documented protective effect of an opaque lens. Individuals with cataracts have a significantly reduced risk of macular degeneration.[3] If the radiation is absorbed in the lens, it cannot possibly harm the retina.

All of the major cellular and molecular features of age-related cataract have been reproduced in the laboratory by radiation in conjunction with heat and oxygen.[1] Moreover, solar radiation can with similar cogency explain the distinctive global pattern of age-related cataract among human populations—the risk of cataract depends on where one lives on the surface of the earth.[1]

Epidemiological evidence represents only a small part of the total evidence implicating sunlight in cataract formation, but it is of particular interest insofar as it has not been reviewed before. A brief sampling of the material will now be presented.

One type of statistic that has been used to measure cataract prevalence is the percentage of blindness due to cataract. When cataract blindness statistics from 55 different countries of the world are grouped according to latitude, it is found that in the tropics there is a fivefold increase in blindness resulting from cataract than at northern latitudes, whereas intermediate latitudes fall in between.[1] Although many factors are involved, sunlight is the only one known to vary in a gradient from high in the tropics to low in the northern latitudes.

Another measure of cataract prevalence is the

number of cataract operations. When the rate of cataract surgery at a northern latitude, in England, is compared with the rates in Israel among immigrants from Europe or from more tropical regions, a gradient with latitude is also revealed. The highest rates of cataract surgery occur in individuals from countries situated nearer to the equator.[1]

In a study of cataract surgery in India, in which latitude was held constant, it was found that individuals who lived in cool, cloudy, and rainy districts had less frequent cataract surgery than those who lived where the rainfall was less, temperature was higher, and sunlight exposure was correspondingly increased.[1]

Direct observations of cataract prevalence obtained from population surveys of 300 000 persons from all over the world also reveal a positive correlation of cataract with latitude. The age-adjusted prevalence of cataract among individuals living in the tropics is twice as high as it is among persons living at northerly latitudes. Cataract prevalence in the intervening zone lies between the two extremes. These results also support the importance of the sunlight factor.[1]

The same association of cataract and sunlight exposure has been disclosed in epidemiological studies where direct measurements of solar radiation have been used. In an investigation of 125 000 people inhabiting different regions of China, the prevalence of cataract was strongly correlated with the local intensity of global radiation—the sum of direct and scattered solar radiation.[1]

Two additional studies largely eliminate the confounding variables of genetics and nutrition. An investigation in Nepal revealed that the prevalence of cataract was positively correlated with the average daily hours of sunlight to which the natives had been exposed during their lives. Similarly, a study of 64 000 Australian aborigines disclosed that in regions where the intensity of solar ultraviolet radiation was greatest, the prevalence of cataract was also highest.[1]

Analysis of the distribution of cataract among human populations leads to the unequivocal conclusion that as exposure to sunlight increases, so too does the prevalence of cataract. This fits perfectly with the other evidence—experimental, biochemical, clinical, and theoretical—implicating sunlight in cataract development. A complete outline of an explanation of this disease is now available.

The sunlight factor is uniquely significant for another reason—we can do something about it. As noted above, reducing ocular exposure to sunlight will diminish the effects of all three major causal factors—heat, oxygen, and solar radiation—in both diseases. This is a conclusion with potentially enormous significance for prolonging the period of visual health.

HIGH-ENERGY COMPONENTS OF SUNLIGHT ARE HAZARDOUS

Sunlight is pure energy radiated from the sun, where it is created by a nuclear fusion reaction. To understand how sunlight acts on the eye, we must refer to the particulate nature of radiation. Sunlight is an incomprehensibly large collection of energy particles called photons, all traveling at the same speed, but differing in their energy content.

The hazardous effects of sunlight on the eye are strictly correlated with the energy content of the sunlight particles. Photons with more energy produce more damage than those with less energy. As the energy increases, so does the damage.

The correlation of photon energy and molecular damage is well-documented in the lens and retina.[1,3] The lens absorbs and is damaged by the highest-energy photons that penetrate the eye. These photons, with energies between 3.1 and 4.0 eV represent the ultraviolet (UV) part of the spectrum. Within the UV range, the amount of lens damage increases as photon energy increases.

The retina is also susceptible to damage from the highest-energy photons that reach it. This has been referred to as "the blue light hazard," since this part of the solar spectrum is interpreted by our brains as being violet and blue. These hazardous photons contain energy that ranges between 2.5 and 3.1 eV.

Those with more energy are more damaging than those with less. Phrased in terms of wavelengths, the lens is damaged by UV radiation ranging between 300 and 400 nm; the retina is at risk from visible radiation between 400 and 500 nm. In both the lens and retina the radiation-absorbing molecules have been identified, and the major pathways of molecular destruction have been revealed.

PROTECTION AGAINST AGE-RELATED EYE DISEASE

Senescence is inevitable, but the rate of aging can be retarded by reducing the effects of the major causes of deterioration. Slowing the rate of decay will delay the onset of age-related eye disease by postponing the transition between aging and disease. Because age-related cataract and macular degeneration are advanced stages of a continuous process of deterioration that begins in childhood, the entire life span is available for

the application of preventive measures to slow the aging process.

Intervention to lower the temperature or to reduce the oxygen content of the human eye is impractical, but it is a simple matter to reduce ocular exposure to the high-energy particles in solar radiation. As described above, this will diminish the amount of damage caused by all three major causal factors in both the lens and retina, producing a sixfold benefit.

It is not necessary to avoid exposure to sunlight to reduce needless radiation damage. The eye can be fully protected from the hazards of solar radiation by the use of appropriate protective lenses—sunglasses of the right kind—that filter out harmful, high-energy photons from sunlight before they can damage the delicate molecular structures of the eye.

PROTECTIVE LENSES

Current evidence provides the basis for the design of protective lenses that minimize the hazards of sunlight exposure without significantly interfering with vision. The prescription has two components—one to protect the lens, the other to protect the retina.

All eyewear, including sunglasses, should block all ultraviolet radiation. This will protect the lens (and the exposed anterior parts of the eye) against radiation damage that accelerates aging. No advantage is gained by exposing any part of the eye to ultraviolet radiation. It is useless for vision in the intact eye and harmful to any part of the eye that absorbs it. There is no more justification for exposing the eye to UV radiation than there is for exposing it unnecessarily to x-rays. All types of eyewear, including prescription lenses for indoor use, contact lenses, and intraocular lens implants, should be rendered impermeable to UV radiation. All optical lens materials currently in use are readily amenable to such treatment either by incorporating (invisible) UV-absorbing molecules into the lens material or by coating the lens with UV-absorbing dye.

Reducing exposure to ultraviolet radiation will diminish the risk not only of age-related cataract, but also of several other forms of ocular deterioration in which UV radiation has been implicated as a causal factor (including pterygium, climatic droplet keratopathy, damage to the corneal endothelium, photokeratitis and photoconjunctivitis, dermatoheliosis, photodermatosis and carcinoma of the conjunctiva and eyelids, intraocular malignant melanoma, and pinguecula). Second, to protect the retina, sunglasses should absorb high-energy visible radiation.

Total absorption of the hazardous "violet and blue" photons will impose a slight color distortion. Consequently, some individuals may prefer to compromise between safety and color perception, by selecting a lens that blocks most but not all of the violet and blue.

In summary, the hazards of exposure to solar radiation can be minimized by the use of protective lenses that absorb all of the UV and most or all of the high-energy visible photons.

Use of sunglasses with these properties should begin early in childhood and be continued throughout the life span whenever exposure to bright sunlight is desirable or necessary. Radiation damage to delicate ocular structures can occur at any age and tends to be cumulative. Even modestly effective preventive measures may produce highly significant benefits if applied over an extended period.

A PROGRAM OF PREVENTION

A program of prevention to reduce the prevalence of age-related ocular disease can be based on the use of protective lenses that absorb the high-energy photons in solar radiation. The public should be informed regarding the problem of age-related eye disease and the available means of protection. Those in the eyecare professions should provide leadership by protecting their own eyes and by explaining to their patients and the public why and how to do it.

This proposal should be acceptable to the American public. The concept of reducing disease by preventive measures is familiar from prior public health campaigns, such as those concerning cigarette smoking, acquired immunodeficiency syndrome (AIDS), and the effects of UV radiation on skin aging and cancer. In addition, the recommended means of prevention is simple, safe, inexpensive, practical, readily available, and easy to understand. Sunglasses interfere negligibly with current lifestyles and already are widely used. Furthermore, sunglasses can be produced in a form that combines protection with style and fashion.

What would be the effect of a 10- to 20-year delay in the onset of age-related cataract? Based on current prevalence rates, a 10-year delay in the transition between aging and disease would reduce the number of elderly persons suffering from cataract by half, sparing 13 to 18 million individuals who would otherwise have been visually impaired. A 20-year delay would reduce the number of afflicted persons to six times fewer than current projections.[1] Similar beneficial results would be achieved by delaying the onset of age-related macular degeneration.

The price of inaction is a future, 50 years from now,

when 50 million elderly Americans will have diminished vision due to cataract and macular degeneration. To go forth knowingly toward that predictable outcome without a plan for prevention, without hope for a medical cure, and without having tested the possible beneficial effects of a simple and safe method of delaying the onset of these diseases seems unwise.

Discovery of the ocular hazards of sunlight is one of the great accomplishments of vision research. It offers the promise of improved visual health through application of the best kind of medicine, preventive medicine.

Author's Note

A list of currently available sunglasses known to offer maximum protection against the high-energy components of sunlight will be supplied on request.

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